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## **A DEVICE WITH A POROUS MEMBRANE FOR IMPROVING CARDIAC FUNCTION**

### **BACKGROUND OF THE INVENTION**

#### **1). Field of the Invention**

[0001] This invention relates to a method and device for improving cardiac function.

#### **2). Discussion of Related Art**

[0002] Congestive heart failure annually leads to millions of hospital visits internationally. Congestive heart failure is a description given to a myriad of symptoms that can be the result of the heart's inability to meet the body's demand for blood flow. In certain pathological conditions, the ventricles of the heart become ineffective in pumping the blood, causing a back-up of pressure in the vascular system behind the ventricle.

[0003] The reduced effectiveness of the heart is usually due to damage to the heart muscle, leading to an enlargement of the heart. A myocardial ischaemia may, for example, cause a portion of a myocardium (of the heart muscle) to lose its ability to contract. Prolonged ischaemia can lead to infarction of a portion of the myocardium wherein the heart muscle dies and becomes scar tissue.

[0004] Once this tissue dies it no longer functions as a muscle and cannot contribute to the pumping action of the heart. When the heart tissue is no longer pumping effectively, that portion of the myocardium is said to be hypokinetic, meaning that it is less contractile than the uncompromised myocardial tissue, or even akinetic. As this situation worsens, the local area of compromised myocardium may in fact bulge out as the heart contracts, further decreasing the heart's ability to move blood forward. When local wall motion bulges out with

each contraction, it is said to be dyskinetic. The dyskinetic portion of the myocardium may stretch and eventually form an aneurysmic bulge. Certain diseases may cause a global dilated myopathy (i.e., a general enlargement of the heart) when this situation continues for an extended period of time. As the heart begins to fail, the filling pressures increase, which stretches the ventricular chamber prior to contraction, greatly increasing pressure (preload) that the heart has to contract against. In response, the heart tissue remodels to accommodate the chronically increased filling pressures, further increasing the work that the now-compromised myocardium must perform.

[0005] This vicious cycle of cardiac failure results in the symptoms of congestive heart failure such as shortness of breath, edema in the periphery, nocturnal dyspnea (a characteristic shortness of breath that occurs at night after going to bed), weight gain, and fatigue, to name a few. The enlargement increases stress on the myocardium. The stress increase requires a larger amount of oxygen supply, which can result in exhaustion of the myocardium leading to a reduced cardiac output of the heart.

#### SUMMARY OF THE INVENTION

[0006] This invention relates to a device for improving cardiac function. The device includes a membrane which is insertable into a ventricle of a heart in a partitioning position, wherein the membrane substantially forms a division between two volumes of the ventricle, namely a first hemodynamic volume and a second relatively hemostatic volume of the ventricle. Blood enters the first hemodynamic volume through an inflow heart valve, circulates through and is contained primarily in the first hemodynamic volume, and leaves the first hemodynamic volume through an outflow heart valve. The membrane

partitioning the first and second volumes confines the passage of blood through the heart to the first hemodynamic volume.

[0007] The membrane has a plurality of fenestrations that are sufficiently large to allow blood from the hemodynamic volume to flow therethrough into the hemostatic volume, for purposes of forming a thrombus in the hemostatic volume. The fenestrations are sufficiently small so as to allow formation of the thrombus within the hemostatic volume by isolating a volume of blood on the static side of the partition and sufficiently reducing flow so as to promote coagulation of that volume of blood. The device thereby predictably and purposefully produces a thrombus contained within the second volume of the chamber.

[0008] The device further includes at least one anchor formation connected to the membrane, the anchor formation having at least one anchoring portion that is positioned and capable of anchoring to tissue of a myocardium of the heart, and so anchor the membrane in the partitioning position to the myocardium.

[0009] The invention also relates to a method for improving cardiac function. A membrane having a plurality of fenestrations is inserted into a ventricle of the heart. The membrane is anchored to a myocardium of the heart in a partitioning position, wherein the membrane substantially forms a division between a first hemodynamic volume of the heart and a second relatively hemostatic volume of the heart. Blood enters the hemodynamic volume through a first respective heart valve, and leaves the hemodynamic volume through a second respective heart valve. The fenestrations are sufficiently large so that blood flows from the hemodynamic volume therethrough into the hemostatic volume, but sufficiently small so that a thrombus grows within the hemostatic volume.

BRIEF DESCRIPTION OF THE DRAWINGS

[0010] The invention is further described by way of example with reference to the accompanying drawings, wherein:

[0011] Figures 1A and 1B are top plan and side views of a frame construction for a device according to an embodiment of the invention, wherein the frame construction is in an open position;

[0012] Figure 2 is a side view of the frame construction in a collapsed condition;

[0013] Figure 3 is a view similar to Figure 2, with partition rim wires of the frame construction being deployed by pulling on a membrane pull wire;

[0014] Figures 4A and 4B are plan and side views of the frame construction, wherein the partition rim wires are expanded by depressing rim wire extenders;

[0015] Figure 5A is a perspective view of a delivery mechanism for transporting and deploying the frame construction;

[0016] Figure 5B is perspective view of the delivery mechanism with an inner shuttle cannula thereof rotated through 180°;

[0017] Figures 6A through 6D are cross-sectional side views through a heart, and Figure 6E is a cross-sectional top plan view through the heart, illustrating deployment of the device within a ventricle of the heart;

[0018] Figures 7 A to 7C are cross-sectional side views illustrating the flow pattern within the ventricle before and after a membrane of the device is deployed within the ventricle;

[0019] Figures 8A to 8D are cross-sectional side views illustrating the use of the membrane to grow a thrombus within the ventricle; and

[0020] Figures 9A and 9B are cross-sectional side and cross-sectional top plan views respectively, illustrating the use of multiple membranes.

#### DETAILED DESCRIPTION OF THE INVENTION

[0021] Figures 1A and 1B illustrate a frame construction 10 of a device according to an embodiment of the invention. Not shown in Figures 1A and 1B is a membrane that is secured to the frame construction 10 and a delivery mechanism used for delivering and deploying the frame construction 10 and the membrane. The frame construction 10 is assembled from seven wires, including an anchor wire 12, two partition rim wires 14, an anchor stern wire 16, two rim wire extenders 18 (Figure 1A only), and a membrane pull wire 20 (Figure 1B only).

[0022] Proximal ends 24 of the partition rim wires 14 are secured to one another, hinged relative to one another, and slidably located on a proximal portion 26 of the anchor wire 12. The rim wire extenders 18 have central portions 30 that extend along the anchor wire 12. Proximal portions 32 of the rim wire extenders 18 are bent outwardly away from the anchor wire 12. Distal ends 34 of the rim wire extenders 18 are secured to the partition rim wires 14. Distal ends 36 of the partition rim wires 14 are secured to one another and to a distal end of the anchor stem wire 16. Ends of the wires 14, 16, and 18 are formed into hooks 40 that can be used for anchoring to tissue of a myocardium. A distal end of the anchor wire 12 is formed into an anchoring screw 42 that can also anchor to a myocardium. Although the hooks 40 and anchoring screw 42 are shown, it should be understood that other anchoring formations may be used, such as clamps, staples, etc.

[0023] As illustrated in Figure 2, the entire frame construction 10 is initially

collapsed, which allows the frame construction 10 to be located within a catheter and to be transported within the catheter into a ventricle of a heart. The anchoring screw 42 can then be turned into a myocardium of the heart, thereby anchoring the distal end of the anchor wire 12 to the myocardium.

[0024] Next, as illustrated in Figure 3, a proximal end of the membrane pull wire 20 is retracted, which pivots the anchor stem wire 16 about its proximal end at the anchor wire 12. Distal ends 36 of the partition rim wires 14, connected to the distal end of the anchor stem wire 16, then move away from the anchor wire 12. The anchoring hook 40 formed by the distal end of the anchor stem wire 16, also separates from the anchoring screw 42 and can be moved into contact with the myocardium at a location distant from the anchoring screw 42.

[0025] As illustrated in Figures 4A and 4B, the proximal ends 32 of the rim wire extenders 30 are then advanced in a direction 44, i.e., toward the anchoring screw 42. The distal ends 36 of the rim wire extenders 30, being connected to the partition rim wires 14, move the partition rim wires 14 outwardly away from one another, with the proximal ends 24 of the partition rim wires 14 sliding along the anchor wire 12 in a direction toward the anchor screw 42.

[0026] Referring again to Figure 1A, the proximal ends 32 of the rim wire extenders 30 can then be rotated in directions 46. Rotation of the rim wire extenders 18 rotates the partition rim wires 14. The partition rim wires 14 are rotated so that they are positioned adjacent to a myocardium in the heart. The anchoring hooks 40 at the end of the rim wire extenders 18 can then also be inserted into the myocardium of the heart. With the anchoring hooks 40 engaged with the myocardium and the partition rim wires 14 located against the myocardium, the anchoring hooks 40 at the proximal ends 32 of the rim wire



extenders 18 can then also be inserted into the myocardium.

[0027] Figure 5A illustrates a delivery mechanism 50 that may be used for deploying the frame construction as hereinbefore described. The delivery mechanism 50 includes an outer delivery catheter sheath 52, an outer shuttle cannula 54, an inner shuttle cannula 56, and an inner guide cannula 58, one coaxially located within the other.

[0028] The outer and inner shuttle cannulas 54 and 56 have key formations 60 and 62 respectively, and retaining openings 66 are formed in the inner shuttle cannula 56. The proximal ends 32 of the rim wire extenders 18 are inserted through the openings 66, thereby securing the rim wire extender 18 to the inner shuttle cannula 56. The anchor wire 12 and the membrane pull wire 20 both extend through the inner guide cannula 58.

[0029] The inner guide cannula 58 initially extends approximately to the anchoring screw 42. Once the anchoring screw 42 is connected to the myocardium, the inner guide cannula 58 is withdrawn into the inner shuttle cannula 56. The membrane pull wire 20 is then retracted to elevate the partition rim wires 14 (as illustrated in Figure 3). The outer and inner shuttle cannulas 54 and 56 are then advanced in a distal direction within the outer delivery catheter sheath 52. Advancement of the outer and inner shuttle cannulas 54 and 56 open the partition rim wires 14 (as illustrated in Figures 4A and 4B). The outer and inner shuttle cannulas 54 and 56 can then be rotated within the outer delivery catheter sheath 52, so that the partition rim wires 14 are rotated (as illustrated in Figures 1A and 1B). The inner shuttle cannula 56 is then partially retracted into the outer shuttle cannula 54 until the proximal ends 32 of the rim wire extenders 18 are partially pushed through the openings 66 by a proximal surface of the key

opening formation 60.

[0030] As illustrated in Figure 5B, the inner shuttle cannula 56 is then rotated within the outer shuttle cannula 54. Anchoring hooks that are located within the inner shuttle cannulas 56 can then be inserted into the myocardium. The inner shuttle cannula 56 can then be retracted so that the proximal ends 32 of the rim wire extenders 18 disengage therefrom. Mechanisms which are known in the art may be used for disengaging the anchor wire 12 and the membrane pull wire 20, whereafter the delivery mechanism 50 can be removed from the heart.

[0031] Figures 6A to 6E illustrate deployment of the device within the left ventricle 160 of a heart 162. As illustrated in Figure 6A, the delivery mechanism 50 is inserted through the aortic valve 164 of the heart 162, and the distal anchoring screw 42 is connected to the myocardium 166 of the heart 162. As illustrated in Figure 6B, the partition rim wires 14 are elevated, and the anchoring hooks 40 at ends thereof are secured to the myocardium 166. As illustrated in Figures 6C and 6D, the delivery mechanism 50 is withdrawn, which leaves the frame construction 10 secured to the myocardium.

[0032] Also forming part of the device is a membrane 170 which has a periphery secured to the partition rim wires 14. The partition rim wires 14 are located on the myocardium 166, so that the membrane 170 forms a division between first and second volumes 172 and 174 of the ventricle 160. Figure 6E illustrates the positioning of the membrane 170 with respect to the aortic valve 164.

[0033] Figure 7A illustrates the flow through the ventricle 160 before an aneurysm is formed out of the ventricle 160. Blood flows through a mitral valve into the ventricle 160, through the ventricle 160, and then exits the ventricle 160

through the aortic valve 164.

[0034] Figure 7B illustrates the flow within the ventricle 160 after an aneurysmic bulge 180 is formed out of the ventricle 160. Small eddy currents 182 are formed within the aneurysmic bulge 180, while flow within an upper portion of the ventricle 160 remains substantially laminar.

[0035] As illustrated in Figure 7C, the membrane 170 forms a division between a hemodynamic volume 172 of the ventricle 160 and a relatively hemostatic volume 170 partially formed by the aneurysmic bulge 180. The blood now flows through the mitral valve into the hemodynamic volume 172, through the hemodynamic volume 172, and then out of the aortic valve 164. The membrane 170 segregates the relatively hemostatic volume 174 from the normal flow area of the heart within the hemodynamic volume 172.

[0036] Figures 8A to 8D illustrate the use of the membrane 170 to create a thrombus. Blood can still pass through fenestrations in the membrane 170 into and out of the hemostatic volume 174. The fenestrations reduce pressures that act on the membrane 170, and so reduce stresses within the membrane 170, the frame construction to which the membrane 170 is mounted, and to the myocardium to which the frame construction 10 is secured. The flow characteristics through the hemostatic volume 174 allow for the growth of the thrombus from the base of the hemostatic volume 174. The thrombus grows to encompass all of the hemostatic volume 174, so as to provide support for the membrane 170. Any remaining stresses within the membrane 170 are thereby substantially diminished. The thrombus shrinks over an ensuing period of time, with the membrane 170 having been rendered inconsequential by having been fully incorporated and endothelialized, forming a new inner wall of the ventricle

160.

[0037] With specific reference to Figure 8A, blood enters from the hemodynamic volume 172 through the fenestrations into the hemostatic volume 174, pressurizes the hemostatic volume 174, and then may flow through the fenestrations back into the hemodynamic volume 174. The fenestrations, by allowing blood to flow therethrough, reduce pressures that act on the membrane 170 and contribute to the predictable propagation of a thrombus within the hemostatic volume 174.

[0038] The flow of blood through the hemostatic volume 174 is slow when compared to circular flow patterns 182 of Figure 7B. The flow within the hemostatic volume 174 near the membrane 170 is primarily turbulent, due to the disruption of flow created by the membrane 170 and the relatively small sizes of the fenestrations. The ongoing flow through the fenestrations in the membrane 170 forestalls the growth of a thrombus on the membrane 170, contributing to the predictable propagation of the thrombus. Additionally, the membrane 170 is preferably thrombolytic to forestall growth of a thrombus on the membrane 170. The flow pattern within a base of the hemostatic volume 174 is primarily turbulent. With reference to Figure 8B, such turbulent flow permits the creation and growth of a thrombus 190 on a base of the hemostatic volume 174. The location of the thrombus 190 does not interfere with flow of blood through the membrane 170 and through the hemostatic volume 174. The fenestrations are large enough so as to allow uncoagulated blood to flow therethrough, but small enough to confine coagulated products of the blood.

[0039] As illustrated in Figures 8C and 8D, the thrombus 190 subsequently grows to fill the entire hemostatic volume 174. The thrombus may then grow by

an additional degree to lift the membrane 170 slightly. The thrombus is then slightly larger than the size of the original hemostatic volume 174. The growth of the thrombus up to the stage of Figure 8D is typically between six and 12 hours, but may be between one hour and thirty days. The thrombus now provides support for the membrane 170, so that pressures created within the hemodynamic volume 172 are transferred through the membrane 170 onto the thrombus 190. Strain within the membrane 170 and forces acting on the myocardium of the heart, which are already low due to the inclusion of the fenestrations, are thereby further reduced to substantially zero.

[0040] A lining on an inner surface of the myocardium of the heart grows to form an endothelial lining over the membrane 170. The endothelial lining forms a wall that ties opposing sides of the myocardium together, and so further assists in absorbing pressures created within the hemodynamic volume 172.

Thrombolytic agents may be applied to the membrane 170 to prevent clot formation, but these thrombolytic agents should not persist for any longer than is necessary to form the thrombus 190, if such thrombolytic agents would prevent endothelialization.

[0041] Over time, the thrombus 190 may begin to shrink, and a portion of the myocardium surrounding the thrombus 190 may begin to recede toward the membrane 170. The hemostatic volume 174 may decrease by at least 20% in twelve months. In order to allow for the thrombus to shrink, the frame construction 10 may at least have portions thereof that are bio-absorbable, or can bend to allow for the myocardium to recede toward the membrane 170.

[0042] It can thus be seen that pressures on the membrane 170 are reduced by providing fenestrations in the membrane 170. The fenestrations also create a

flow pattern within the hemostatic volume 174, which promotes growth of the thrombus 190. The growth of the thrombus 190 is promoted from a base of the hemostatic volume 174 toward the membrane 170, so that the thrombus 190 eventually lifts the membrane 170 and then forms the primary structure which further absorbs pressures from the hemodynamic volume 172.

[0043] It is believed that at least some of the fenestrations in the membrane 170 should be at least 30 microns wide to allow for a sufficient blood flow rate therethrough. It is also believed that none of the fenestrations should be larger than 1000 microns, preferably no larger than 500 microns, so as to create the correct flow pattern within the hemostatic volume 174 and promote the creation and growth of the thrombus 190. The fenestrations preferably form between 25% and 75% of the entire area of the membrane 170, and there are typically at least 1000 of the fenestrations.

[0044] The membrane 170 is preferably made of a material which is non-thrombogenic. Materials that are non-thrombogenic may include Nomex™, Kevlar™, Spectra™, PET, PTFE, PGA, PLA, PEEK, Nylon™, Nitino™, stainless steel Eligiloy™, gold-plated molybdenum, or platinum. In order to be bio-absorbable, the membrane 170 may be made of a material such as PGA, PLA, pericardium, or small intestinal submucosa. These materials are all intervascularly biocompatible.

[0045] Figure 9A illustrates the use of multiple devices, each having a respective membrane 170A-C. The orientations of the devices are such that different locations of hypokinetic segments of the heart can be isolated. Figure 9B illustrates the positioning of two membranes 170D and 170E in an overlapping manner as viewed from a mitral valve annulus.

[0046] While certain exemplary embodiments have been described and shown in the accompanying drawings, it is to be understood that such embodiments are merely illustrative and not restrictive of the current invention, and that this invention is not restricted to the specific constructions and arrangements shown and described since modifications may occur to those ordinarily skilled in the art.

CLAIMS

What is claimed:

1. A device for improving cardiac function, comprising:

a membrane which is insertable into a ventricle of a heart, in a partitioning position wherein the membrane substantially forms a division between a hemodynamic volume and a relatively hemostatic volume of the ventricle, blood entering the hemodynamic volume through an inflow heart valve, circulating through the hemodynamic volume and leaving the hemodynamic volume through an outflow heart valve, the membrane having a plurality of fenestrations that are sufficiently large to allow blood from the hemodynamic volume to flow therethrough into the hemostatic volume, for purposes of forming a thrombus in the hemostatic volume, but sufficiently small so as to allow formation of the thrombus within the hemostatic volume; and

at least one anchor formation connected to the membrane, the anchor formation having at least one anchoring portion that is positioned and capable of anchoring to tissue of a myocardium of the heart, and so anchor the membrane in the partitioning position to the myocardium.

2. The device of claim 1, wherein each fenestration is between 30 and 1000 microns wide.

3. The device of claim 2, wherein each fenestration is less than 500 microns wide.

4. The device of claim 2, wherein there are at least 100 of the fenestrations.

5. The device of claim 2, wherein the fenestrations form between 25% and 75% of an area of the membrane.



6. The device of claim 2, wherein the membrane does not have a fenestration that is larger than 1000 microns.
7. The device of claim 1, wherein at least surfaces of the membrane facing the hemodynamic volume are thrombolytic.
8. The device of claim 1, wherein the membrane has at least 100 fenestrations, each of which is between 30 and 500 microns, the fenestrations forming between 25% and 75% of an area of the membrane, and at least surfaces of the membrane facing the hemodynamic volume being non-thrombogenic.
9. The device of claim 1, wherein the membrane is made from an intravascularly biocompatible material.
10. The device of claim 9, wherein the material is selected from the group consisting of Nomex™, Kevlar™, Spectra™, PET, PIFE, PGA, PLA, PEEK, Nylon™, Nitinol™, stainless steel, Eligiloy™, gold-plated molybdenum, and platinum or other suitable material in conventional use.
11. The device of claim 1, wherein the membrane is made from a bio-absorbable material.
12. The device of claim 11, wherein the material is selected from the group consisting of PGA, PLA, pericardium, and small intestinal submucosa.
13. The device of claim 11, wherein each anchoring formation has a sharp end to penetrate the myocardium.
14. The device of claim 1, further comprising:  
a frame construction, including a rim to which the membrane is

peripherally attached, an anchor wire to which the rim is connected, and a pull wire connected to the rim for moving a portion of the rim away from the anchor wire.

15. The device of claim 14, comprising at least one anchor formation on the anchor wire and at least one anchor formation on the rim.

16. A device for improving cardiac function, comprising:  
a rim which is movable from a collapsed state into an open state;  
a pull wire connected to the rim, retracting of the pull wire moving a distal portion of the rim;  
an anchor formation on the rim; and  
a membrane having a periphery secured to the rim.

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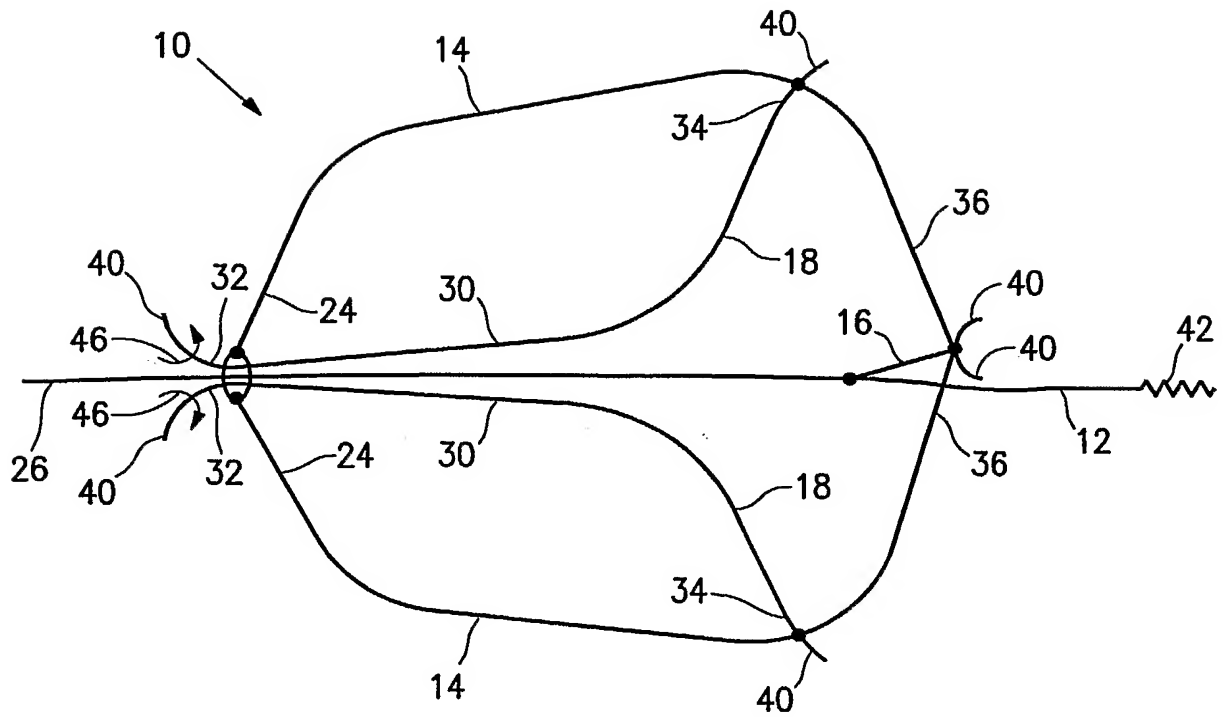


FIG. 1A

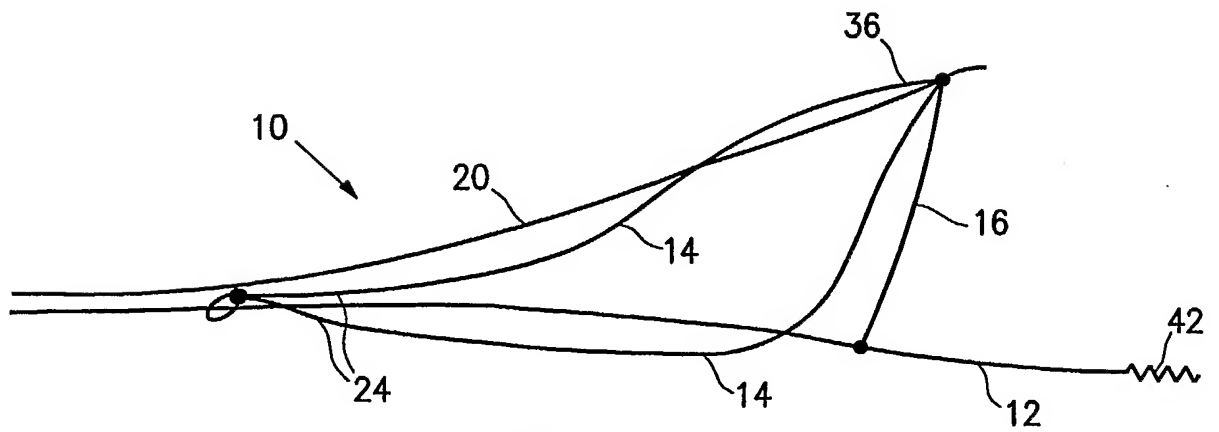


FIG. 1B

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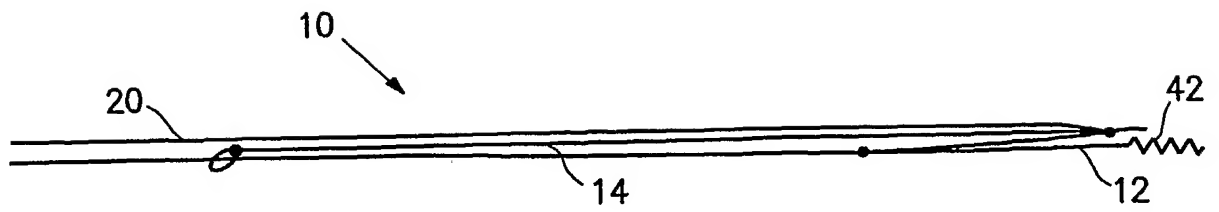


FIG. 2

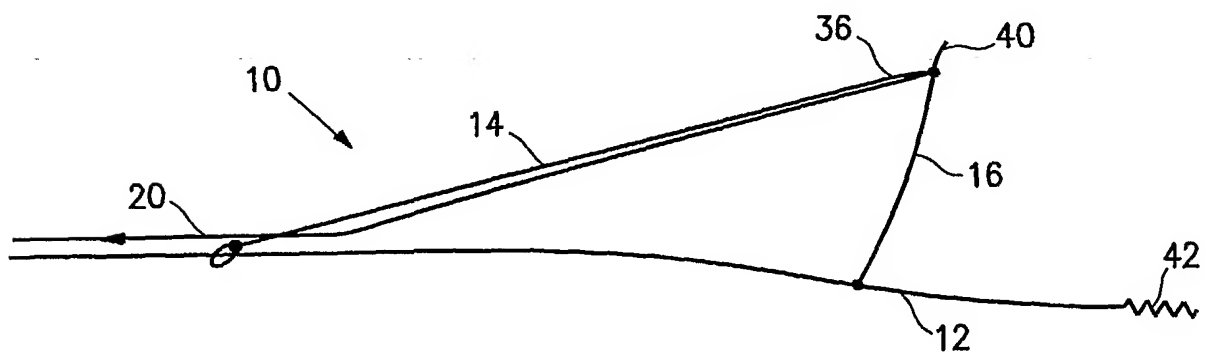


FIG. 3

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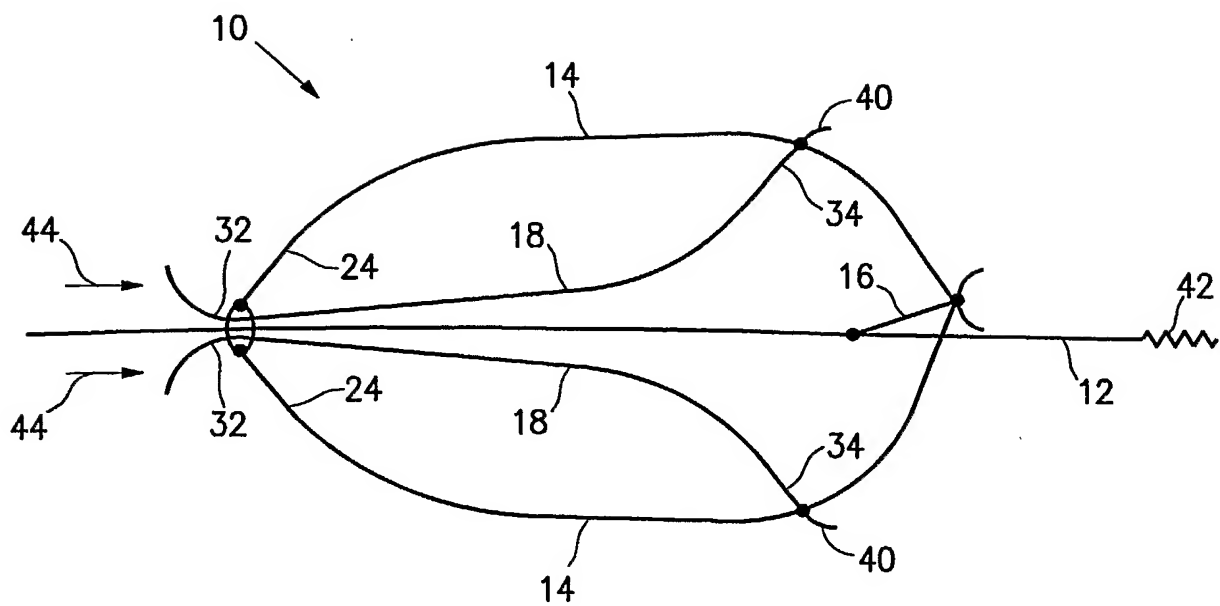


FIG. 4A

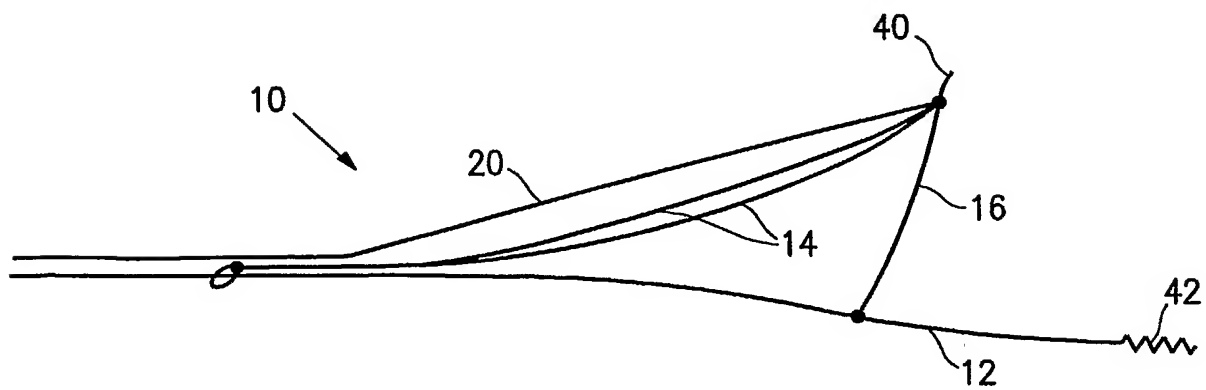


FIG. 4B

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FIG. 5A

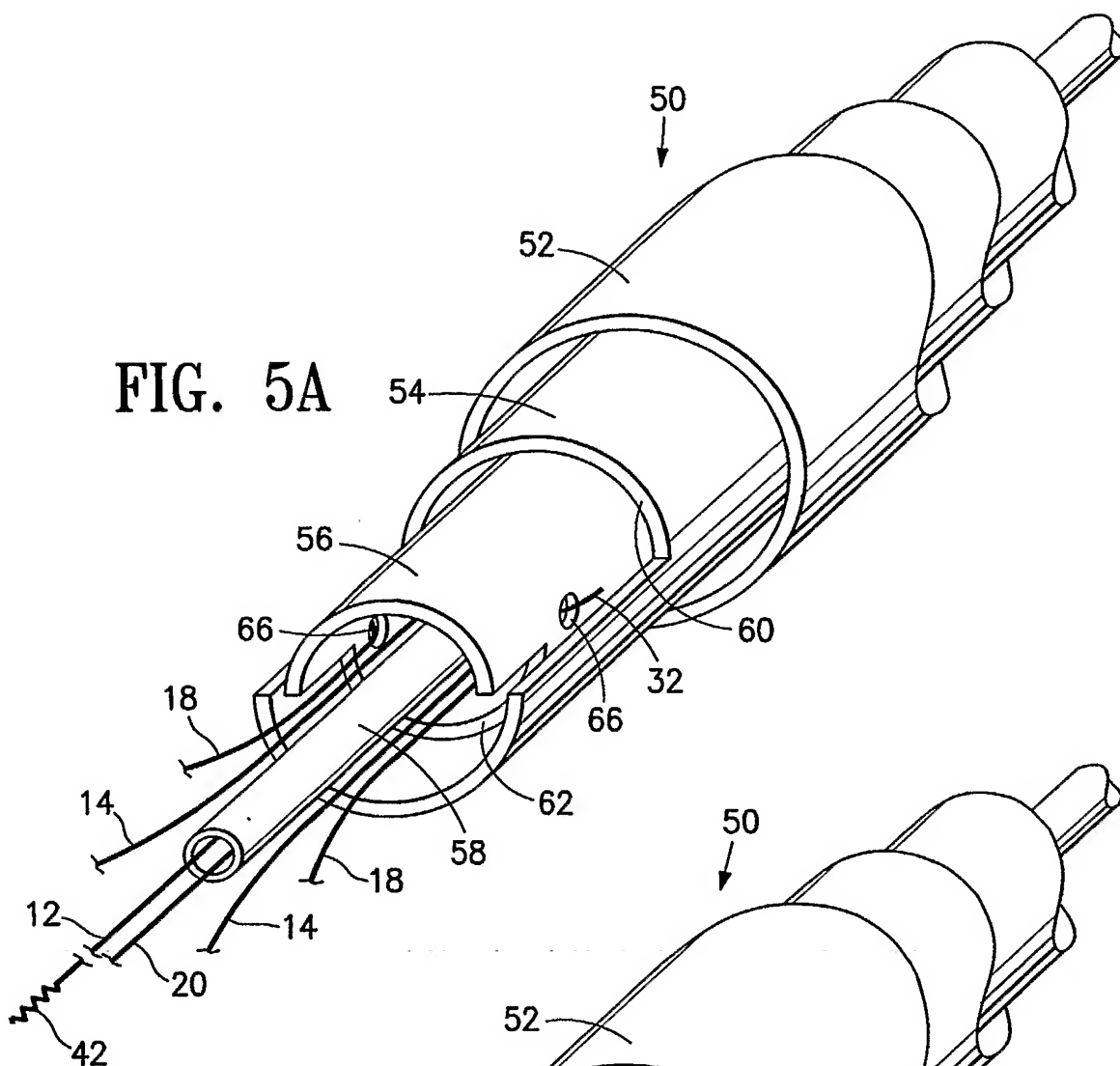
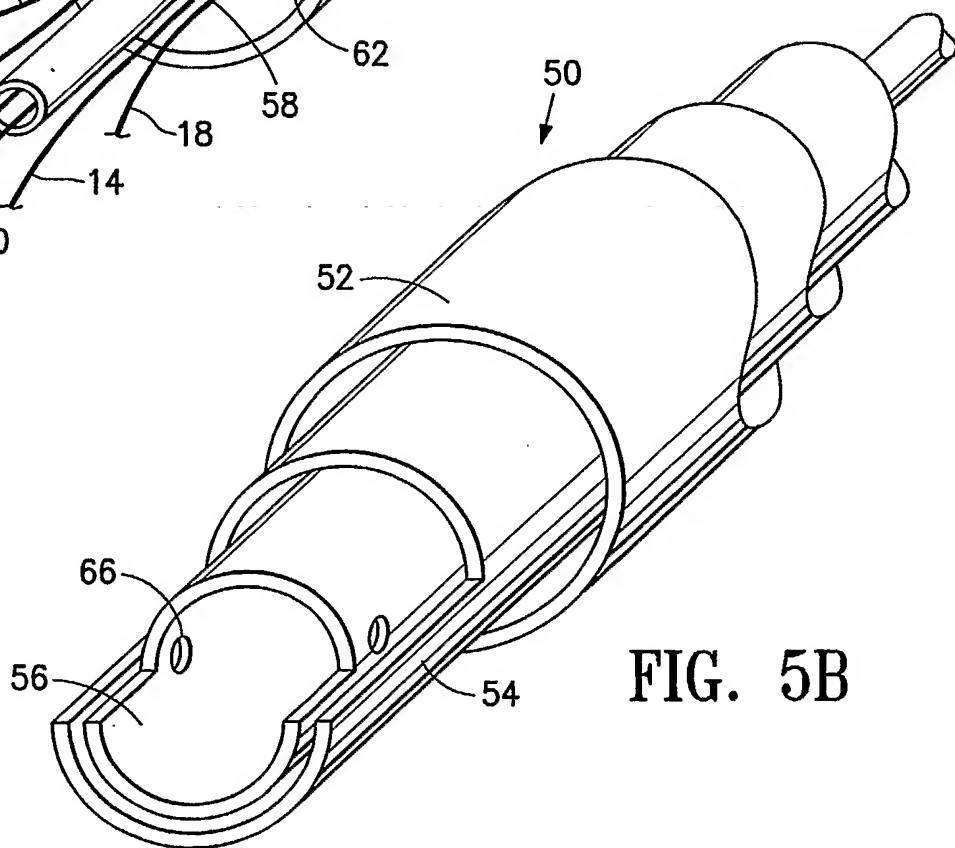


FIG. 5B



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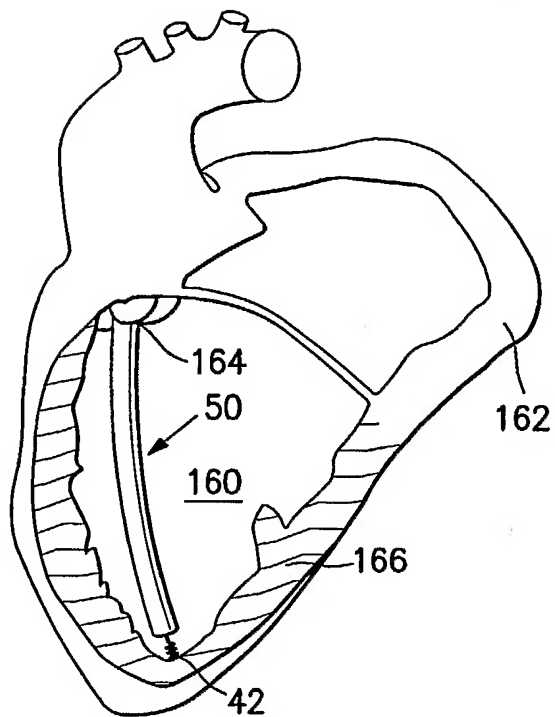


FIG. 6A

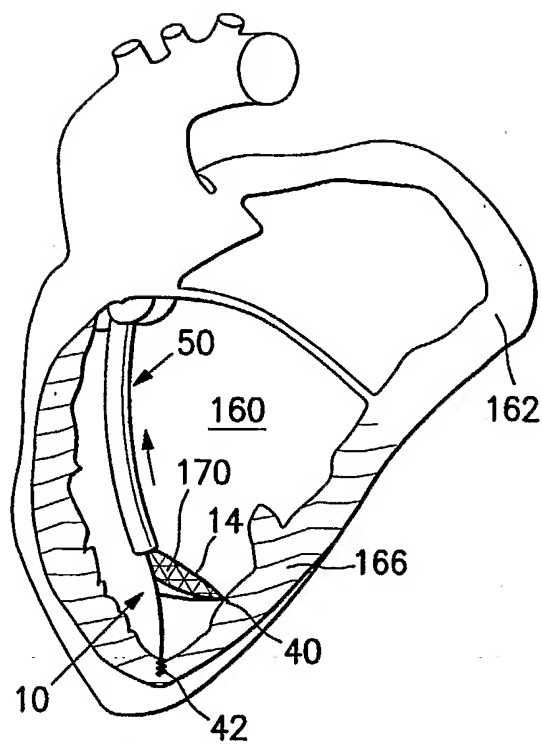


FIG. 6B

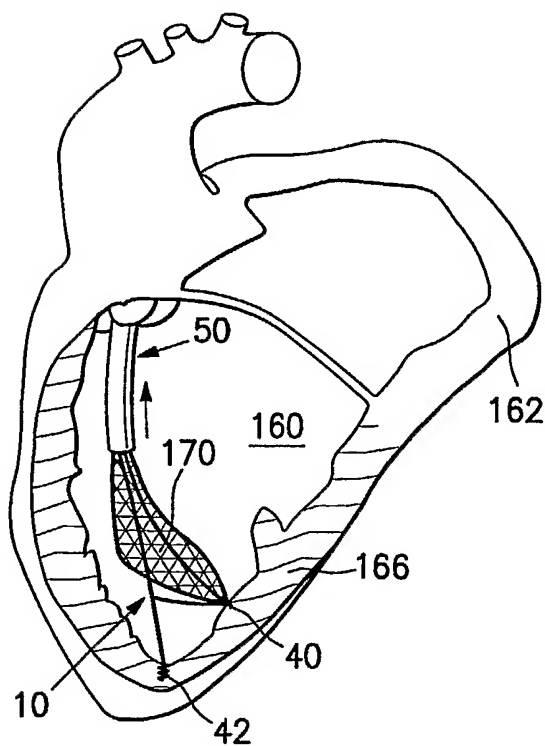


FIG. 6C

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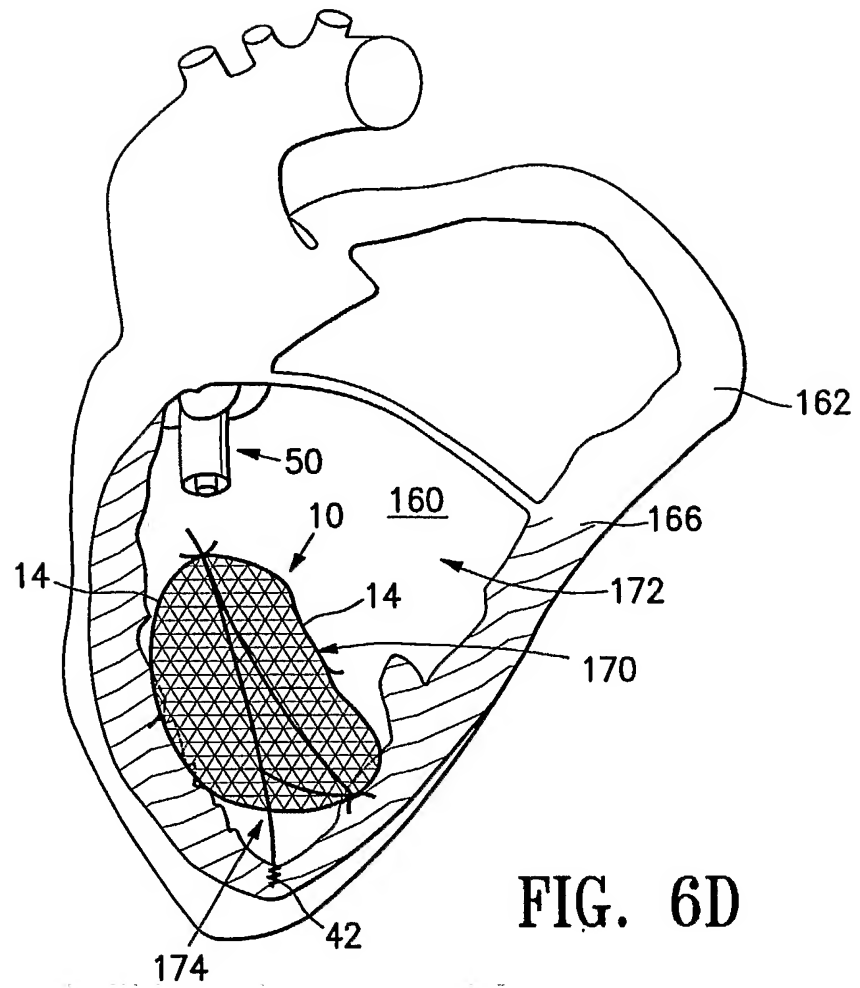


FIG. 6D

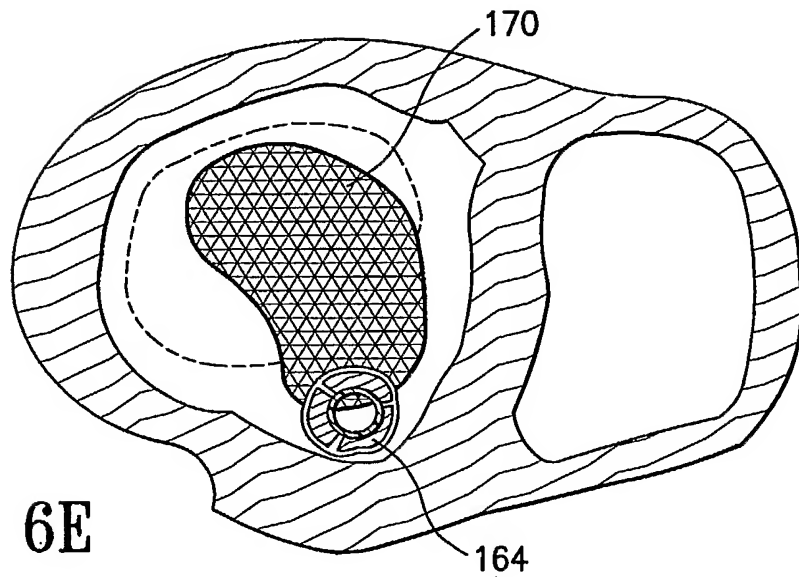


FIG. 6E



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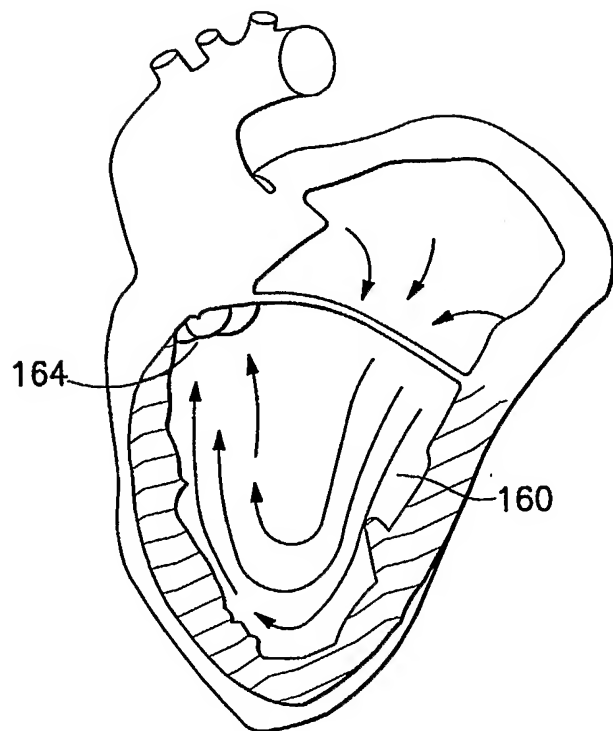


FIG. 7A

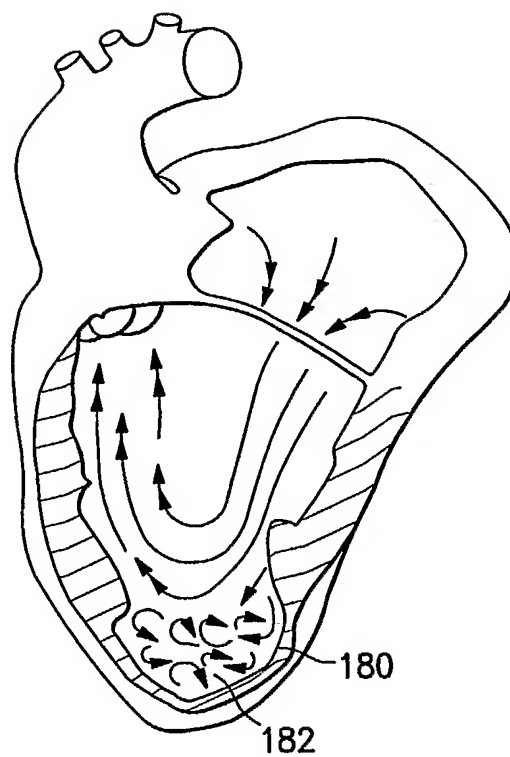


FIG. 7B

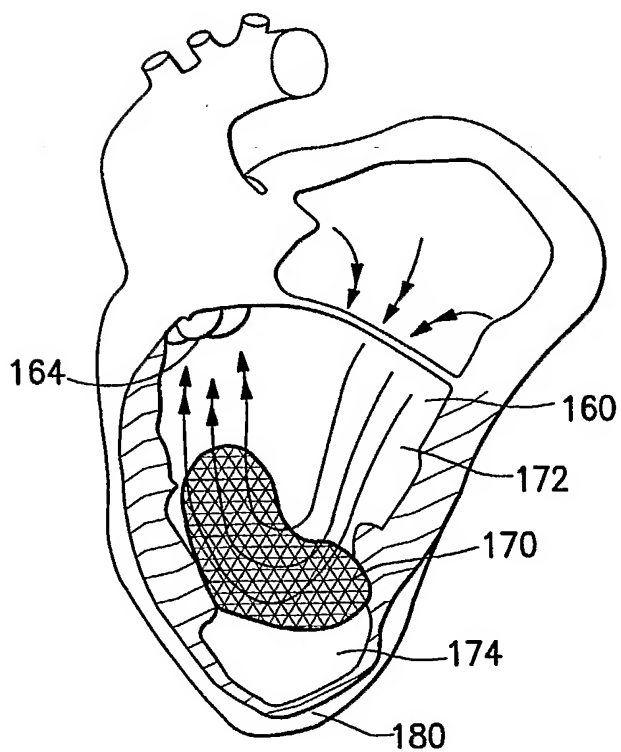


FIG. 7C

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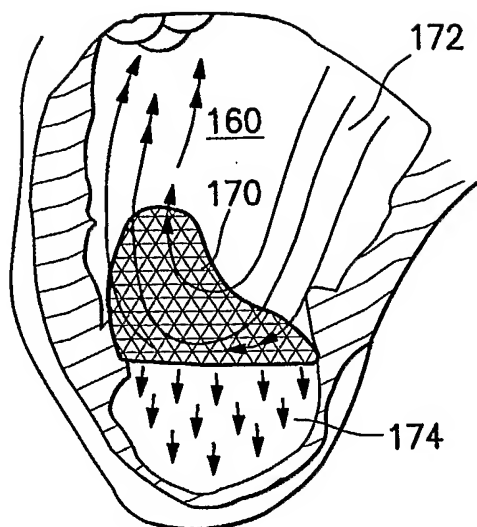


FIG. 8A

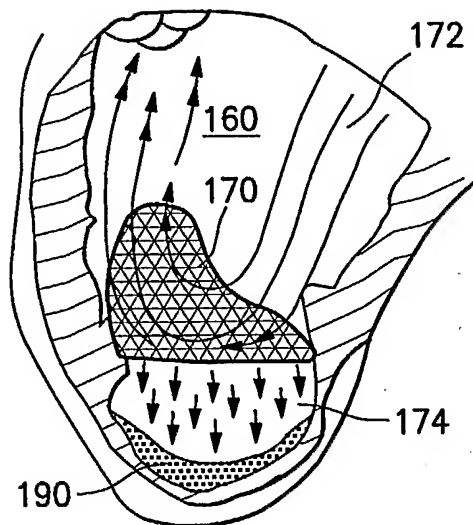


FIG. 8B

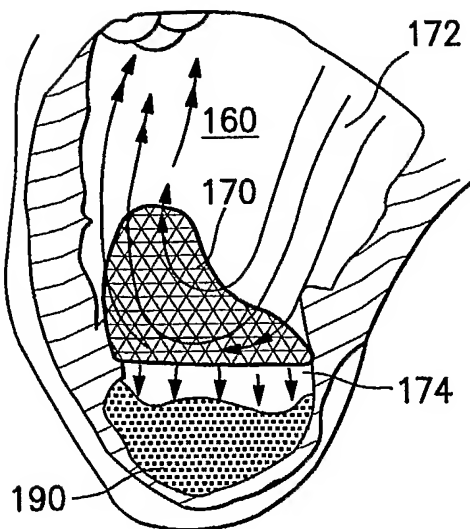


FIG. 8C

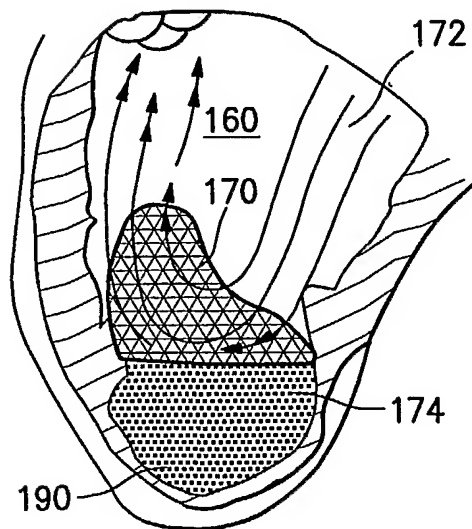
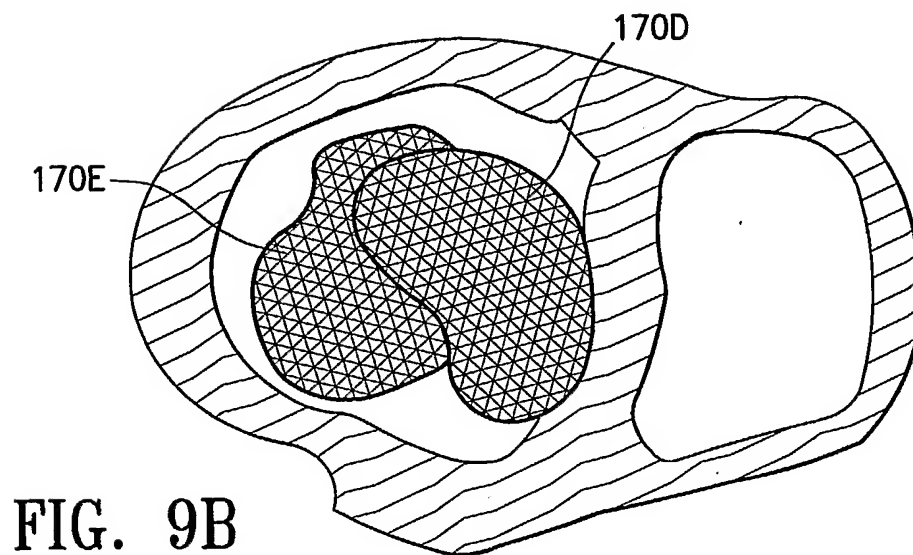
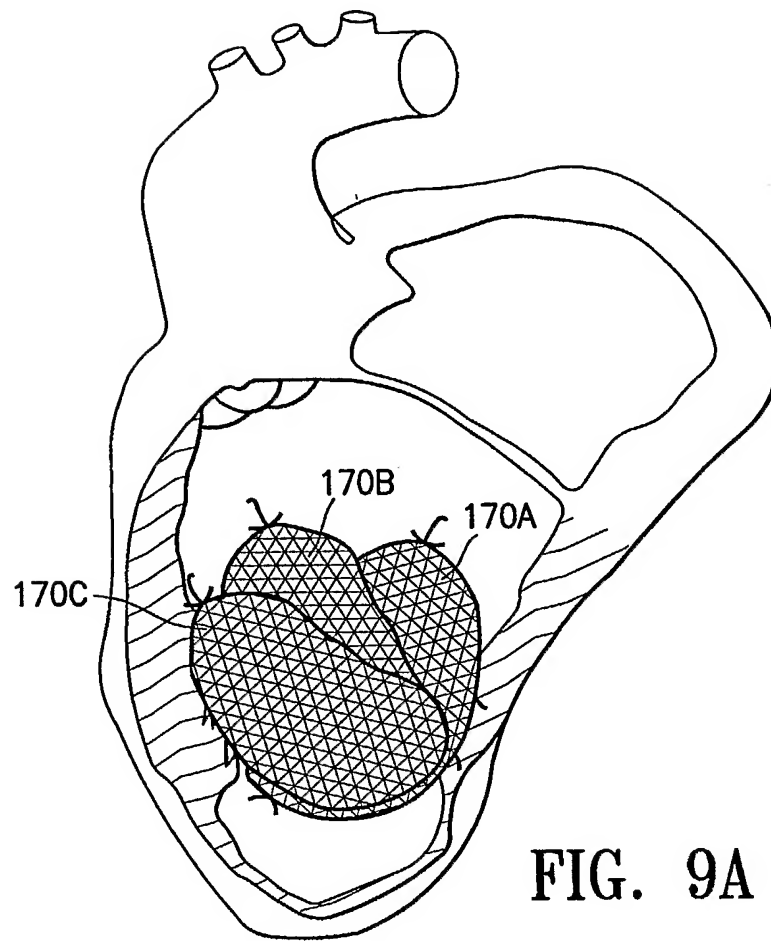


FIG. 8D

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# INTERNATIONAL SEARCH REPORT

Int lional Application No

PCT/US 03/36864

**A. CLASSIFICATION OF SUBJECT MATTER**  
IPC 7 A61F2/00

According to International Patent Classification (IPC) or to both national classification and IPC

## B. FIELDS SEARCHED

Minimum documentation searched (classification system followed by classification symbols)

IPC 7 A61F

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched

Electronic data base consulted during the international search (name of data base and, where practical, search terms used)

EPO-Internal

## C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category °	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
E	WO 2004/012629 A (SEPTUS INC) 12 February 2004 (2004-02-12) paragraph '0023! - paragraph '0048! -----	1-16
A	US 2002/169359 A1 (KEITH PETER T ET AL) 14 November 2002 (2002-11-14) paragraph '0198! - paragraph '0205! -----	1,16
A	US 5 961 440 A (SCHWEICH JR CYRIL J ET AL) 5 October 1999 (1999-10-05) column 5, line 46 - column 10, line 34 -----	1,16
P,A	WO 03/007778 A (FELD YAIR ; RELAXIS LTD (IL)) 30 January 2003 (2003-01-30) page 26, line 5 - page 49, line 30 -----	1,16
P,A	US 2003/149333 A1 (ALFERNESS CLIFTON A) 7 August 2003 (2003-08-07) paragraph '0019! - paragraph '0030! -----	1,16

☐ Further documents are listed in the continuation of box C.

☒ Patent family members are listed in annex.

° Special categories of cited documents:

\*A\* document defining the general state of the art which is not considered to be of particular relevance

\*E\* earlier document but published on or after the International filing date

\*L\* document which may throw doubts on priority claim(s) or which is cited to establish the publication date of another citation or other special reason (as specified)

\*O\* document referring to an oral disclosure, use, exhibition or other means

\*P\* document published prior to the international filing date but later than the priority date claimed

\*T\* later document published after the international filing date or priority date and not in conflict with the application but cited to understand the principle or theory underlying the invention

\*X\* document of particular relevance; the claimed invention cannot be considered novel or cannot be considered to involve an inventive step when the document is taken alone

\*Y\* document of particular relevance; the claimed invention cannot be considered to involve an inventive step when the document is combined with one or more other such documents, such combination being obvious to a person skilled in the art.

\*S\* document member of the same patent family

Date of the actual completion of the international search

5 April 2004

Date of mailing of the international search report

14/04/2004

Name and mailing address of the ISA

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